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# Autism as a Contingency-Shaped Disorder of Verbal Behavior: Evidence Obtained and Evidence Needed

**Michael D. Hixson**
**Central Michigan University**

Drash and Tudor's argument that autism is a contingency-shaped disorder of verbal behavior is logical and consistent with behavioral principles, but the argument's premises have no direct empirical support and some conflicting evidence. The quantity and quality of research needed to support such a theory is compared to that found in the area of antisocial behavior in children, which has considerable evidence for a contingency-shaped etiology. Even if autism is largely inherited, this does not weaken the necessity or importance of behavioral intervention. Drash and Tudor's paper may serve a useful function by outlining areas in need of further study because a great deal more research is needed on how the early environment shapes the language, cognitive, and behavioral development of children.

Environmental/behavioral theories of the etiology of behavior disorders seem to be increasingly rare as popularity grows for genetic and other biological explanations. Barkley (1997), for example, discusses the evidence for neurological and genetic causes of attention deficit hyperactivity disorder but says "there have been few efforts to articulate a purely environmental cause for ADHD in children." (p. 88). The decline in behavioral explanations is not due to just the advancements in genetics but also to an apparent lack of interest in etiology from a behavioral perspective. For most of the behavioral disorders discussed in the *International Handbook of Behavior Modification and Therapy* (Bellack, Hersen, & Kazdin, 1990), there is no discussion of etiology. Few theories of the etiology of behavior disorders can be found in the behavioral literature and the few that do exist are not often cited. Perhaps the increased interest in functional behavior assessment, which addresses the function of specific classes of behavior, has, maybe appropriately, reduced interest in finding the etiology of broad-based classes of behavior disorders.

Autism is commonly accepted as a neurological disorder in the medical field (NIMH, n.d.; Veenstra-Vandedrweele & Cook, 2003) and among psychologists (e.g., Kaufman, 2001; Newsom & Hovanitz, 1997). As for behavior analysts, a popular behavior analysis Web site says, "Autism and the other disorders in the autism spectrum are behaviorally defined syndromes that are now generally regarded to be of neurobiological origin." (Cambridge Center for Behavioral Studies, n.d.) The NIMH and private organizations, such as Cure Autism Now (CAN), have committed millions of dollars to finding the biological cause or causes of autism. The CAN organization is providing resources to help identify the genetic and environmental causes of autism, but "environment" in this case refers to the neurotoxicity of mercury and its possible role in autism—not the learning environment. Within this affable climate for biological etiologies and scarcity of behavioral etiologies comes Drash and Tudor's theory of autism as a contingency-shaped disorder, which concludes that attributing autism to biological variables is unnecessary.

Drash and Tudor's argument is not so much based on the weakness of the biological evidence, but rather on the logic of their theory and the strength of the behavioral research. The brief discussion of neurobiological causes of autism correctly points out that no definitive biological causes have been identified, and that there are no reliable biological markers for diagnosis. The most reliable diagnostic systems are those that assess behavioral dimensions (American Psychological Association, 2001). However, Drash and Tudor do not address the evidence for the apparent heritability of autism. A monozygotic (identical) twin has an approximately 60% chance of meeting the criteria for autism, while a dizygotic (fraternal) twin has approximately the same chance as other sib-

Address correspondence to Michael D. Hixson, Ph.D., Psychology Department, Central Michigan University, Mt. Pleasant, MI 48859; phone: (989) 774-6462; e-mail: hixso1md@cmich.edu.

lings, which is 4.5% (Veenstra-Vanderweele & Cook, 2003).

Drash and Tudor describe four premises on which their theory is based. While each of the premises is likely to have a direct impact on a child's development, the link between each of these variables and autism is not direct. In other words, no experiments are described that directly link the variables discussed in the premises to children diagnosed with autism. The first premise is that a lack of reinforcement contingencies for the development of a verbal repertoire is responsible for the verbal behavior deficit in children with autism. The research by Hart and Risley (1995, 1999) is cited in support of this point, but no research is described that supports the role of these variables in children with autism. There is evidence that contradicts this premise. Mothers of children with autism engage in the same frequency of verbal interaction as mothers of language-matched control children who do not show the other social and behavioral characteristics of autism (Cantwell, Baker, & Rutter, 1977). It would probably not be useful to compare the frequency of parent-child verbal interactions of families with a child with autism to families with only normally developing children because it is known that mothers change the frequency and type of verbal interaction in response to the language skills of the child (Moerk, 1974). Also, in Hart and Risley's correlational research, the genetic variables are perfectly confounded with the parenting practices, which make this research unlikely to convince behavioral geneticists (Rowe, 1994). Research is needed that identifies functional relations between parenting practices and child development by experimental manipulation.

The second premise is that caregivers may inadvertently shape disruptive and avoidance repertoires early in a child's life. Drash and Tudor cite the research of Gewirtz and Pelaez-Nogueras, which found that disruptive infant behavior could be inadvertently shaped by parents. No direct research evidence of the role of this variable in autism is provided. As is the case with the frequency of verbal interactions, there is evidence that contradicts this point. Parents of children with autism interact with their child in a way similar to that of parents of children with language delays but not the other features of autism (Cantwell, Baker, Rutter, 1979).

The third premise is that disruptive behavior is often present in infants and children with autism. The evidence consists of the authors' extensive clinical practice with children with autism. The fourth premise is that these disruptive behaviors interfere with the acquisition of appropriate verbal behavior. This possible developmental sequence requires further empirical evidence. While there is evidence that acquiring a particular behavioral repertoire can hinder or facilitate the acquisition of further repertoires, this is an area sorely in need of additional research (Hixson, in press).

In short, the quality and quantity of research in support of Drash and Tudor's theory is limited. The reinforcement paradigms described by Drash and Tudor that may lead to the repertoires found in children with autism are logical and consistent with behavioral principles. However, direct experimental findings are needed. What might this research consist of? The quality and quantity of research needed might best be explained by exemplification.

There may be only one behavior disorder in which there is convincing evidence for a learning etiology. The evidence is strong enough that many introductory and abnormal psychology textbooks describe it as a possible cause of the disorder. The behavior disorder is antisocial behavior in children and adolescents. Patterson and colleagues at the Oregon Social Learning Center (OSLC) have conducted the most extensive research in this area (Reid, Patterson, & Snyder, 2002). The research program began in the 1960s with the discovery that intervention methods based on the experimental analysis of behavior were more effective than the psychodynamic methods that were popular at that time. An extensive line of research on children with behavior problems demonstrated that changing the contingencies by changing parent and teacher behavior produced large changes in child behavior. Such findings suggested that early experiences in the home might be responsible for the initial development of the antisocial repertoire.

Patterson and colleagues hypothesized that coercive interaction patterns in homes of children with behavior problems were responsible for the child's deviant behavior (Patterson, 1982). The coercive pattern consists of the use of negative reinforcers to shape the behavior of family members. In particular, a parent direction followed by an aversive reaction by the

child often resulted in the parent terminating the direction, thereby reinforcing the child's aversive behavior. Once the parent terminated her direction, the child stopped emitting the aversive behavior, which reinforced the parent's giving in. The theory was supported by experiments conducted in clinical laboratories in which the reactions of the parents were experimentally manipulated, but whether these reaction patterns actually occurred in homes was still unknown. Next, Patterson and colleagues conducted observations of parent-child interactions in the home in which interaction sequences were carefully coded. The findings further supported the theory that negative reinforcement played a key role in the development of antisocial behavior (Reid, Patterson, & Snyder, 2002).

A number of other variables have been found to correlate with child and adolescent antisocial behavior, such as living in a low-income home, having divorced parents, low performance in school, and peer rejection. Patterson and colleagues provided strong evidence that low-income, divorce, and similar "stressor" variables were only related to child antisocial behavior when they disrupted parenting practices (Patterson, Reid, & Dishion, 1992). The poor school performance was due to the effectiveness of the antisocial repertoire in avoiding academic tasks and the poor control that adult instructions had on the children's behavior. This type of extensive line of research conducted at OSLC is what would be needed to support Drash and Tudor's theory.

Drash and Tudor do not discuss the possibility of an interaction between biological and environmental variables. Rutter (1997) discusses the many ways in which genetic and environmental variables may interact. In the case of antisocial behavior, the findings from one study suggest that early child management difficulties may produce poor parenting practices (Stoolmiller, 2001). A child's predisposition and parenting practices would then work synergistically to produce an antisocial repertoire. Rutter makes the important point that even something that has a high heritability estimate may be quite amenable to environmental intervention. This is because the behavioral effects of genetic variables are influenced in their expression by the environment. Stated differently, heritability estimates are based on common or current environmental arrangements; if the environments are changed substantially, the heritability estimate may change. The environment that applied behavior analysts expose children with autism to when doing intensive behavioral treatment is one such substantial change in environmental contingencies.

In conclusion a great deal more research is needed to be confident in Drash and Tudor's theory. With no studies demonstrating an actual difference in parenting practices, the theory appears speculative. Even though the empirical evidence presented by Drash and Tudor is limited, it would be a mistake to casually dismiss the theory. The factors discussed by Drash and Tudor would logically produce the behavioral repertoire seen in autism and children with language delays in general. While the evidence for the role of these variables in autism is limited, it is possible that these variables may play a role in some cases of autism or other language and cognitive disorders. Whatever the etiology of autism, Drash, Tudor, colleagues, and many other behavior analysts have demonstrated the remarkable power of behavioral techniques in many areas of child development, such as autism recovery (Lovaas, 1987), prevention of functional retardation (Bijou, 1981), remediation of mental retardation (Drash & Leibowitz, 1973), remediation of language delays in preschoolers (Drash & Tudor, 1989), acceleration of cognitive development in normal infants (Drash, 1990 as described in Storfer, 1990), and cognitive improvement in children with Down syndrome (Drash, 1982). I think it is likely that this intervention research and Drash and Tudor's wide-ranging clinical experience, more so than the cited studies, has influenced their thinking on the etiology of autism. But further evidence for a neurobiological cause of autism and related disorders does not reduce the importance of behavioral methods. As previously discussed, even if autism was found to be highly heritable, this does not mitigate the necessity for behavioral intervention.

Hopefully, Drash and Tudor's paper will stimulate research on the effects of parenting practices on the cognitive and language development of children. The field of child development needs to be studied from an experimental analysis of behavior perspective (e.g., Staats, 1971). We know very little about the cumulative effects of the learning principles or how learning one repertoire affects later learning,

but research on just such things is needed to understand the development of complex human behavior from a learning perspective.

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